What is claimed is:

1. A chimeric fusion protein comprising an amino terminus and a carboxyl terminus, wherein the protein comprises insulin chain B and individual peptide moieties consisting of and at least one GAD 65 peptide(s) capable of eliciting a human T cell response, wherein the insulin chain B and the at least one human GAD 65 peptide(s) are covalently linked and the chimeric fusion protein is capable of eliciting a human T cell response to insulin chain B and to each of the at least one GAD 65 peptides.

2. The chimeric fusion protein of Claim 1, wherein the GAD 65 peptide is selected from the group consisting of human GAD 65 peptides 115-127, 247-286, and 473-519.

3. The chimeric fusion protein of Claim 2, wherein the protein comprises, in order, starting at the amino terminus, human GAD 65 peptides 115-127, 247-286, and 473-519.

4. The chimeric fusion protein of Claim 3, wherein the protein comprises, in order, starting at the amino terminus, insulin chain B, insulin chain C and human GAD 65 peptides 115-127, 247-286, and 473-519.

5. The protein of claim 1 wherein the protein does not contain an amino acid sequence comprising the sequence of amino acid residues 139-173 of SEQ ID NO:2.

6. The chimeric fusion protein of claim 1, wherein, when tested in an assay in a mouse model of IDDM, the chimeric fusion protein provides a greater reduction in the frequency of onset of diabetes than a control mixture containing equimolar amounts of each of the various

comprised by the chimeric fusion protein, each of said individual peptide moieties and insulin chains not being covalently bound to any other of said individual peptide moieties and insulin chains in said mixture, wherein the assay is carried out by the repeated parenteral administration of a number of measured doses, each dose being of a predetermined molar quantity of said chimeric fusion protein in a pharmaceutically effective carrier or of said control mixture in the pharmaceutically effective carrier, the administration being at intervals of not less than twelve hours and not more than 72 hours between each of the doses.

7. The chimeric fusion protein of Claim 6, wherein the mouse model of IDDM is the NOD mouse CYTOXAN induced diabetes model.

8. The chimeric fusion protein of Claim 6, wherein the mouse model of IDDM is the NOD/SCID mouse adoptive transfer diabetes model.

9. A chimeric fusion protein comprising the amino acid sequence spanning amino acid residues 2 to 154 of SEQ ID NO:1.

10. A chimeric fusion protein comprising the amino acid sequence spanning amino acid residues 2 to 174 of SEQ ID NO:2.

11. A chimeric fusion protein comprising the amino acid sequence spanning amino acid residues 2 to 138 of SEQ ID NO:3.

12. A chimeric fusion protein comprising the amino acid sequence spanning amino acid residues 2 to 175 of SEQ ID NO:4.

13. A chimeric fusion protein comprising the amino acid sequence spanning amino acid residues 2 to 226 of SEQ ID NO:5.

14. A chimeric fusion protein comprising the amino acid sequence spanning amino acid residues 2 to 387 of SEQ ID NO:6.

15. A chimeric fusion protein comprising the amino acid sequence spanning amino acid residues 2 to 438 of SEQ ID NO:7.

16. A method of treating a patient selected from the group consisting of patients predicted to be at risk of developing Type 1 diabetes and patients suffering from Type 1 diabetes, said method comprising the repeated administration of a dose of a measured amount of the polypeptide of any one of Claims 1-15 in a pharmaceutically effective carrier to said patient.

17. The method of Claim 16 wherein the repeated administration comprises administration of at least two doses at an interval of not less than twelve hours and not more than 72 hours.

18. A deletion mutant GAD 65 polypeptide wherein the polypeptide is at least 10 kDa in mass and the sequence spanning amino acid residues 140-172 of SEQ ID NO:2 has been deleted.